

reproduce the complexity of arborization. We used the larval class IV sensory neuron in *Drosophila* as the model cell to approach this question.

As class IV neurons display self-similarity over a range scales, the first key morphological parameter we use to study them is their fractal dimension. The fractal dimension of a neuron is a measure of its complexity and has been used to distinguish between classes of neurons. The second morphological parameter of a neuron involves realizing that such a branching structure can be viewed as a binary tree in which neuronal branching points are the nodes. The structure of interest here is the distribution of node depths, where the depth of a node is the number of other nodes between it and the root (i.e., the cell body) on the tree.

Using both analytical techniques and *in silico* simulations, we made three findings. 1) The fractal dimension was always a monotonically increasing function of the neuron's maximal depth. 2) The observed Gaussian node-depth distributions are achievable via a termination rule in which the probability of branch termination is a sigmoidal function of node depth. 3) The observed node-depth distributions can be qualitatively accounted for by an "inheritance rule", whereby each daughter segment inherits morphological information from its mother segment.

In conclusion, we demonstrate that a set of statistical rules accounts for the fractal dimension and node-depth distribution of class IV neurons.

4005-Pos Board B733

Statistical Constraints on Dendritic Branching Morphology in *Drosophila* Class IV Sensory Neurons

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The morphology of a neuron is key to its function, but the principles that govern neuronal morphogenesis are not clear. To investigate these principles, we used the larval class IV sensory neuron in *Drosophila* as a model cell. Class IV neurons have highly branched dendritic morphology. Our specific question is whether this branching morphology arises from purely random processes or whether there exist non-random constraints on morphological parameters such as segment lengths and branching angles.

To measure the statistical characteristics of the dendritic arbors, we imaged class IV neurons by confocal microscopy and analyzed their skeletons using Fiji and Matlab. First, we found that the lengths of dendritic segments, both terminal and non-terminal, followed exponential distributions. Given that the lengths of the dendritic segments are defined by consecutive branch points, this observation suggests that branching events follow a spatial Poisson process. Second, we found that the angles between two daughter segments follow a normal distribution with a mean of 96 degrees and a standard deviation of 31 degrees ($n = 465$). Because the mean differs from 180 degrees, we conclude that the branching angles are not uniformly distributed. These properties, namely the distributions of segment lengths and angles, were observed throughout morphogenesis.

Our results indicate that there are morphological properties of class IV neurons which are not determined by purely random processes.

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Mismatch Between the Resting Membrane Potential and the Voltage at Maximum Amplification in Outer Hair Cells (OHCs) of Mammalian Cochlea

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OHCs amplify sound by an electromechanical mechanism. Sound-induced vibrations cause OHC membrane potential (E) to change from its resting voltage (E_m) to a new value ($E_m + dE$). The induced receptor potential (dE) initiates charge movement (Q) and force production to counteract viscous losses incurred by the traveling wave. Q exhibits a sigmoidal function with E and because it is most sensitive to dE at the midpoint (V_m), E_m should equal V_m to ensure maximum amplification. V_m was measured with isolated OHCs extracted from guinea pig with whole-cell patch clamp under constant intracellular pressure in presence of KCNQ4 blocker XE991 ($\geq 30 \mu\text{M}$) to ensure robust voltage clamp (conductance $< 1.5 \text{ nS}$ at V_m). After correcting for physiological conditions the results show V_m is coincident with *in vitro* measurements of E_m (Neuron 2011 70: 1143), but a mismatch of 40 mV is apparent when comparing with *in vivo* measurements of E_m made at basal (J. Physiol. 1987 383: 551 and Proc. R. Soc. Lond. B. 1992 247: 97) or apical (J. Neurosci. 1985 5: 1591) regions of cochlea. Results also reveal variation of V_m across the cochlea as a function of a non-uniform charge density of the lateral wall (σ); when σ is uniform V_m is constant, and when σ varies inversely with area of lateral wall (A_{LW}) V_m increases monotonically from a hyperpolarized

value at the high frequency region of cochlea to a depolarized value at low frequency region. Although the relationship between V_m and σ is satisfying as it reflects the electric field, the disparity between *in vitro* and *in vivo* measurements highlights the need to reconcile them to ascertain the operating position of the amplifier.

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Remodeling of the Postsynaptic Density: A Macromolecular Signaling Complex

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The postsynaptic density (PSD), a macromolecular protein machine that resides under the postsynaptic plasma membrane, regulates the efficiency of synaptic transmission by stabilizing neurotransmitter receptors in the membrane and organizing signaling molecules within the postsynaptic compartment. Data suggests that synaptic activity results in changes in the protein composition of the PSD and it is hypothesized that these changes lead to structural modifications that explain enduring and stable alterations in synaptic function. However, direct evidence for these structural changes has never been obtained and the extent of remodeling and the mechanisms responsible are not fully understood. Our long term goal is to create a high-resolution molecular model of the PSD that accurately represents the number and 3D relationships between its protein components allowing hypothesis to be generated about how recruitment or loss of specific proteins results in structural alterations at the PSD. The ubiquitin proteasome system (UPS) targets proteins for degradation and is, in part, responsible for modifications of the proteins that compose the PSD. Synaptic activity has been shown to induce proteasomal recruitment into the postsynaptic compartment, that requires prior activity-dependent recruitment of CaMKII, resulting in changes in protein composition of PSDs. Electron cryotomography (ECT) and immunogold labeling were employed to examine the 3D structure of isolated PSDs and to identify scaffold molecules targeted by the UPS. Proteasome levels were found to be highest in PSDs isolated earlier in development, providing evidence that the UPS plays a crucial role in the structural reorganization of PSDs. ECT will also be utilized to examine whether CaMKII functions as a direct scaffold for the proteasome that might serve as a mechanism to spatially restrict protein degradation.

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Action Potential Collision in Nerves

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It is generally accepted that the collision of two action potentials coming from opposite directions is produced by the mutual annihilation of both signals. The experimental confirmation of this effect was shown by Tasaki in 1949 [1] and their findings are conforming to the Hodgkin-Huxley model for action potential propagation [2].

In the current work we performed an analogous experiments to these made by Tasaki but using *Lumbricus terrestris* as an animal model. The collision of two simultaneously generated impulses propagating in orthodromic and antidromic directions were investigated. The experiments have been performed in the extracted ventral cord of *Lumbricus terrestris* by using double external stimulation and single channel recording. Surprisingly, the collision of two action potential impulses of orthodromic and antidromic propagation within the median giant axon in the ventral cord haven't showed the annihilation of the two signals as is commonly known. The results are in a good agreement with the soliton model for the nerve signal propagation suggested by Heimburg and Jackson [3].

References

- [1] Tasaki I., "Collision of two nerve impulses in the nerve fibre" *Biochimica et Biophysica Acta* 3 (1949) 494-497.
- [2] Hodgkin A.L. and Huxley A.F., "A quantitative description of membrane current and its application to conduction and excitation in nerve. *J. Physiol.* (1952) 177, 500-544.
- [3] Heimburg T. and Jackson A.D., "On soliton propagation in biomembranes and nerves. *Proc. Natl. Acad. Sci. USA.* 102 (2005) 9790-9795.

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Modeling and Simulations of Biomechanical Symptoms of Parkinson's Disease

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Patients with Parkinson's disease experience oscillatory motion of body parts (tremor) due to an increased reaction time. The tremor is an early-stage